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water) should be applied. Under no circumstances should any dressing be allowed to remain on a vaccination wound longer than 24 hours, and no dressing should be applied so long as the natural protection is intact.

On account of possible fouling by perspiration and to lessen the chance of exposure to street dust, primary vaccination should be performed preferably in cool weather.

In order to encourage proper surgical treatment, no charge should be made for the aftercare of a vaccination nor for revaccination in case the first attempt should prove unsuccessful.

Although apparently trivial, vaccination is an operation which demands skill in performance and care in aftertreatment in order to avoid the rare, but serious, complications. For the prevention of these complications vaccination (*a*) should be performed with strictly aseptic technique, (*b*) should cover the smallest possible area for each insertion, and (*c*) should be treated without any covering which permits maceration.

A child should be vaccinated by the time it has reached the age of 6 months, and the operation should be repeated at about 6 years of age and whenever an epidemic of smallpox is present.

WINTER OUTBREAK OF POLIOMYELITIS.

ELKINS, W. VA., 1916-17.

By J. P. LEAKE, Passed Assistant Surgeon; JOSEPH BOLTEN, Passed Assistant Surgeon; and H. F. SMITH, Assistant Surgeon, United States Public Health Service.

Particular attention was attracted to the West Virginia epidemic of poliomyelitis because it occurred in the winter and because it followed so closely the New York epidemic as to be really an extension of that general epidemic. At the time it was thought to be the first winter outbreak in the United States; indeed it was the first to assume anything like epidemic proportions, but a search of the records has revealed other outbreaks in this country during cold weather. Scandinavian observers had called attention to a few winter outbreaks in Sweden and Norway although no definite study had been made of the temperature conditions. A criterion must be adopted as to what constitutes a winter outbreak; the mere months of the year signify very different weather in different parts of the globe. Further, we know that occasional poliomyelitis occurs practically everywhere and in all seasons; these isolated winter cases, however, are unimportant as regards a study of conditions of infection, for it has been known ever since the epidemiologic study of poliomyelitis began that some cases occur apparently without any relation to each other, just as do cases of noninfectious nervous diseases. Therefore it is here assumed that to be considered a winter outbreak, two or more connected cases must occur after the temperature has remained below freezing for 24 hours. A study of the available literature reveals 38 such outbreaks (Table 1), not including the West Virginia epidemic. The official meteorological records were studied, Norwegian,[1] Swedish,[2] Austrian,[3] Prussian,[4] Hessian,[5] or American,[6] as the case might be, and by interpolation from the surrounding stations, the approximate temperatures of the places where outbreaks occurred could be obtained. Besides mere distance, there had to be taken into consideration altitude, proximity to bodies of water, and direction of mountain ranges. Fortunately there were meteorological stations at several of the places themselves, as at Elkins.

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[1] Jahrbuch des Norwegischen meteorologischen Instituts: Kristiania.

[2] Meteorologiska Iakttagelser i Sverige: Uppsala & Stockholm.

[3] Jahrbücher der k. k. Zentral-Anstalt für Meteorologie und Geodynamik: Wien.

[4] Veröffentlichungen des Königlichen Preussischen Meteorlogischen Instituts von G. Lüdeling: Berlin.

[5] Deutsches Meteorlogisches Jahrbuch, Grossherzogtum Hessen: Darmstadt.

[6] Records of the U. S. Weather Bureau, published and unpublished.

TABLE 1.—*Winter outbreaks of poliomyelitis.*

No.	Date.	Place.	Number of cases.	Latitude.	Reference.	Temperature notes.
1	Nov. 24 to Dec. 7, 1886	Christiania, Norway	2	60° N.	Leegaard,[1] p. 154	Maximum, 10° C. (50° F.), 17 days before first case. Minimum, – 9° C. (16° F.), 4 days before last case.
2	November to December, 1904	Aa, Norway	4	64° N.	Leegaard,[1] p. 165	Maximum, 8° C. (46° F.). Minimum, –17° C. (1° F.). Below freezing every day save 6 during 2 months.
3	February to March, 1905	Misvär, Norway	7	67° N.	Leegaard,[1] p. 170	Maximum, 3° C. (37° F.). Minimum, –34° C. (–29° F.). Below freezing entire time. Maximum below freezing during entire 2½ months save 10 days.
4	....do	Sandbäcken estate, Sweden.	3	58° 45′ N.	Wickman,[2] p. 155	Maximum, 7° C. (45° F.). Minimum, –20° C. (–4° F.). The first outbreak of the great 1905 Swedish epidemic.
5	March and April, 1905	Söndres Valdres, Norway.	7	60° 40′ N.	Leegaard,[1] p. 167	Maximum, 6° C. (43° F.). Minimum, –22° C. (–8° F.). Below freezing every day save 2.
6	March, 1905	Östvaagö, Norway	4	68° 15′ N.	Leegaard,[1] p. 171	Maximum, 5° C. (41° F.). Minimum, –8° C. (18° F.).
7	October, 1905, to spring, 1906	Hadsel and vicinity, Norway.	45	68° 15′ N.	....do	Maximum, 8° C. (46° F.). Minimum, –10° C. (14° F.). Part of an intense epidemic in a sparsely settled region 120 miles north of the Arctic circle.
8	October to November, 1905	Odensäker, Sweden	4	58° 45′ N.	Wickman,[2] p. 163	Maximum, 10° C. (50° F.). Minimum, –16° C. (3° F.).
9	March, 1906	Skjerstad, Norway	6	67° N.	Leegaard,[1] p. 177	Maximum, 3° C. (37° F.). Minimum, –23° C. (–9° F.).
10	Oct. 1 to Dec. 20, 1905	Carlshamn group, Sweden.	9	56° 10′ N.	Wickman,[2] p. 216	Maximum, 14° C. (57° F.). Minimum, –11° C. (12° F.).
11	Oct. 2 to Dec. 7, 1905	Storsjö group, Sweden	18	58° 10′ N.	Wickman,[2] pp. 211-213	Maximum, 9° C. (48° F.). Minimum, –14° C. (7° F.).
12	November, 1905, to Feb. 2, 1906	Åtvidaberg group, Sweden	40	58° 20′ N.	Wickman,[2] pp. 241-244	Maximum, 6° C. (43° F.). Minimum, –20° C. (–4° F.). Intense epidemic, apparently direct contagion between cases.
13	Nov. 14 to Dec. 3, 1905	Medåker, Sweden	3	59° 30′ N.	Wickman,[2] p. 234	Maximum, 7° C. (45° F.). Minimum, –12° C. (10° F.).
14	Dec. 14 and 17, 1905	South Vesterbotten, Sweden.	2	64° 15′ N.	Wickman,[2] p. 261	Maximum, 4° C. (39° F.). Minimum, –11° C. (12° F.).
15	November and December, 1905	Hörnsjö, Sweden	4	63° 55′ N.	Wickman,[2] p. 260	Maximum, 5° C. (41° F.). Minimum, –16° C. (3° F.).
16	Nov. 19 to 23, 1905	Mellanset, Sweden	7	63° 30′ N.	Wickman,[2] p. 259	Maximum, 6° C. (43° F.). Minimum, –15° C. (5° F.).
17	November, 1908, to February, 1909	Vienna and Lower Austria	125	48° 15′ N.	Zappert,[3] p. 64	Maximum, 9° C. (48° F.). Minimum, –11° C. (12° F.).
18	Nov. 20 to Dec. 3, 1909	Marburg and suburbs, Germany.	10	50° 50′ N.	Müller,[4] pp. 66, 67	Maximum, 9° C. (48° F.). Minimum, –11° C. (12° F.).
19	December, 1909	Hager and vicinity, Germany.	41	51° 20′ N.	Krause,[5] p. 148	Maximum, 12° C. (54° F.). Minimum, –5° C. (23° F.).
20	November to December, 1909	Central Nebraska	20	41° N.	McClanahan,[6] p. 1161	Maximum, 27° C. (81° F.). Minimum, –26° C. (–14° F.).
21	January to March, 1910	Jämtland, Sweden	25	63° N.	Wickman; translated by Maloney,[7] p. 101.	Maximum, 7° C. (45° F.). Minimum, –30° C. (–22° F.).
22	Oct. 10, 1910, to Mar. 17, 1911	Mo, Norway	15	66° 15′ N.	Leegaard,[1] p. 180	Maximum, 8° C. (46° F.). Minimum, –20° C. (–4° F.).
23	December, 1910, to Jan. 1, 1911	Ware, Mass	3	42° 15′ N.	Rosenau, Sheppard & Amoss,[8] p. 748.	Maximum, 9° C. (48° F.). Minimum, –19° C. (2° F.).
24	Mar. 16 to Apr. 11, 1911	Mosjöen, Norway	17	66° N.	Leegaard,[1] p. 205	Maximum, 13° C. (55° F.). Minimum, –18° C. (–0.4° F.). Direct contact between several of the cases.
25	October to December, 1911	Norrbotten, Sweden	18	65° –69′ N.	Wernstedt,[9] p. 241	Maximum, 11° C. (52° F.). Minimum, –23° C. (–9° F.).
26	Nov. 2 to Dec. 12, 1911	Elverum, Norway	11	60° 50′ N.	Leegaard,[1] p. 191	Maximum, 13° C. (55° F.). Minimum, –18° C. (–0.4° F.).
27	Mar. 15 to Apr. 15, 1911	Färila, Sweden	7	61° 45′ N.	Wernstedt,[9] p. 237	Maximum, 11° C. (52° F.). Minimum, –25° C. (–13° F.).
28	April, 1911	Hotagen, Sweden	3	64° N.	Wernstedt,[9] p. 239	Maximum, 12° C. (54° F.). Minimum, –20° C. (–4° F.).

TABLE 1.—*Winter outbreaks of poliomyelitis*—Continued.

No.	Date.	Place.	Number of cases.	Latitude.	Reference.	Temperature notes.
29	Apr. 1 to 15, 1911	Tjäl, Sweden	5	63° 50′ N	do	Maximum, 9° C. (48° F.). Minimum, −15° C. (5° F.).
30	Feb. 2 to Mar. 26, 1911	Aa, Norway	8	64° N	Leegaard,[1] p. 200	Maximum, 6° C. (43° F.). Minimum, −13° C. (9° F.).
31	January and February, 1911	Levanger, Norway	12	63° 50′ N	Leegaard,[1] p. 201	Maximum, 6° C. (43° F.). Minimum, −21° C. (−6° F.).
32	October, 1911, to January, 1912	Rollag, Norway	16	60° N	Leegaard,[1] pp. 188–189	Maximum, 11° C. (52° F.). Minimum, −17° C. (1° F.).
33	November, 1911, to January, 1912.	Senjen Island and vicinity, Norway.	20	69° 30′ N.; over 200 miles north of Arctic Circle.	Leegaard,[1] p. 207	Maximum, 8° C. (46° F.). Minimum, −13° C. (9° F.).
34	October, 1911, to Feb. 10, 1912	Kolvereid, Norway	25	64° 45′ N	Leegaard,[1] p. 204, fig. 27	Maximum, 10° C. (50° F.). Minimum, −15° C. (5° F.).
35	Jan. 5 to Feb. 20, 1912	North of Mandal, Norway.	9	58° 15′ N	Leegaard,[1] p. 197	Maximum, 6° C. (43° F.). Minimum, −21° C. (−6° F.).
36	November, 1911, to Mar. 8, 1912	Göteborg, Sweden	54	57° 40′ N	Wernstedt,[9] pp. 240 and 242	Maximum, 11° C. (52° F.). Minimum, −18° C. (−0.4° F.).
37	Nov. 21 to Dec. 14, 1916	Lansdowne, Pa	5	39° 55′ N	Le Boutillier,[10] p. 206	Maximum, 18° C. (64° F.). Minimum, −5° C. (23° F.).
38	March, 1917	Waterbury, Vt	5	44° 20′ N	Public Health Reports[11]	Maximum, 13° C. (55° F.). Minimum, −24° C. (−11° F.).
39	Nov. 22, 1916, to Feb. 11, 1917	Randolph, Taylor, and Marion Counties, W. Va.	[12] 78	39° N	Personal investigation and information furnished by Dr. Weirich, of the West Virginia Department of Health.	Maximum, 21° C. (70° F.). Minimum, −23° C. (−10° F.).

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[1] Leegaard, Dr. Chr.: 1914–15—Deutsch. Ztschr. f. Nervenhlk., vol. 53.

[2] Wickman, Dr. Ivar: 1907—Beiträge zur Kenntnis der Heine-Medinschen Krankheit, Berlin.

[3] Zappert, J.: 1911—Studien über die Heine-Medinschen Krankheit: Leipz. u. Wien.

[4] Müller, Eduard: 1910—Die spinale Kinderlähmung: J. Springer, Berlin.

[5] Krause, Paul: 1911—Die akute epidemische Kinderlähmung: Therap. d. Gegenwart, vol. 52, 145–152, 208–214.

[6] McClanahan, H. M.: 1910—A brief report of the Nebraska epidemic of poliomyelitis: J. Am. M. Ass., vol. 55, 1160.

[7] Maloney, W. J. M. A.: 1913—Translation of Wickman: Acute Poliomyelitis. Monograph Series No. 16: J. Nerv. & Ment. Dis. Publishing Co., N. Y.

[8] Rosenau, Sheppard & Amoss: 1911—Boston Med. and Surg. J., vol. 164, 743–748.

[9] Wernstedt: 1912—Some epidemiological experiences from the epidemic of infantile paralysis in Sweden in 1911: Report from the State Medical Institute of Sweden: Stockholm.

[10] Le Boutillier: 1917—Am. J. M. Sc., vol. 153, 188–206. (Feb.)

[11] Public Health Reports, Apr. 20, 1917: Vol. 32, p. 574.

[12] For the purpose of comparison with other epidemics, only the paralytic cases occurring during cold weather are here counted, 36 in Elkins, 22 in Grafton, 15 in Fairmont, and 5 rural. Of the recognized nonparalytic cases during the epidemic period there were in Elkins 34, in Grafton 6, in Fairmont 4, and 2 rural, making a total of 124 for this group of foci.

It is to be seen that some of these outbreaks were far north of the Arctic Circle. In some, the thermometer went below freezing nearly every day of the outbreak, and in some it remained below practically the entire time. In 15 of the outbreaks the thermometer went below zero Fahrenheit, the lowest temperature recorded being 29° below zero Fahrenheit—34° below zero Centigrade—at Misvär, Norway. The contagion was apparently of the most direct sort in a few cases, notably in some of a group south of Åtvidaberg, Sweden, and in the Mosjöen epidemic in Norway. The infrequent communication between houses in cold weather, and the general infrequence of sporadic, unrelated cases at this season, make it possible to trace contact between individual cases much more definitely than in summer, so that the absence in most instances of clear histories of exposure to recognized cases, as we shall see in studying the West Virginia outbreak, is all the more significant in showing the great importance of unrecognized cases and of carriers in the spread of the disease.

Origin of the Elkins Epidemic.

The general origin of the Elkins outbreak was undoubtedly the great New York epidemic of 1916.

It was shown at the Hygienic Laboratory that the West Virginia and the New York diseases were the same, by finding monkeys which had had an attack of poliomyelitis from the Elkins virus immune to the New York virus.

No direct connection could be traced to previous epidemics as regards the cases themselves, but it seems probable that the highly infective virus reached Elkins through Somerset County, Pa., and Garrett County, Md., in each of which a sharp outbreak occurred during the summer and autumn of 1916, the Maryland cases being on the whole later than those in Pennsylvania. From Somerset County into Garrett County, and from Garrett County down to Elkins, there is a highway, an automobile road, which carries much local traffic. The cases in Garrett County were strikingly distributed along this highway, and not in accordance with the general population distribution. No early cases were discovered along the road north of Elkins, but much of the automobile travel from Elkins goes directly into Maryland without stopping at intermediate points. One or more unrecognized cases or healthy carriers would therefore be assumed to be responsible for the Elkins outbreak.

Of the small outbreaks occurring in the United States during the summer of 1917, five were in localities within or adjoining West Virginia, i. e., those occurring in Rockingham and Page Counties, Va.; Allegany and Garrett Counties, Md.; Marion County, W. Va.; Belmont County, Ohio; and Newcastle, Pa.

In a house-to-house canvass of Elkins, search was made for old cases of poliomyelitis; that is, cases with onsets in previous years. Nine such were found in a population of 6,340. No relation of these cases to the origin of the epidemic could be made out. At least eight other old cases of paralytic disease in children were found which were not poliomyelitis. Of the nine poliomyelitis cases, four had their onset in Elkins and five were in persons living elsewhere at the time. The ages at onset were as follows:

	Cases.		Cases.		Cases.
Under 1 year	0	2 years	1	4 years	1
1 year	4	3 years	2	19 years	1

The years of onset were as follows:

	Cases.		Cases.		Cases.
1900	2	1905	2	1910	1
1901-1903	0	1906-1908	0	1911	2
1904	1	1909	1		

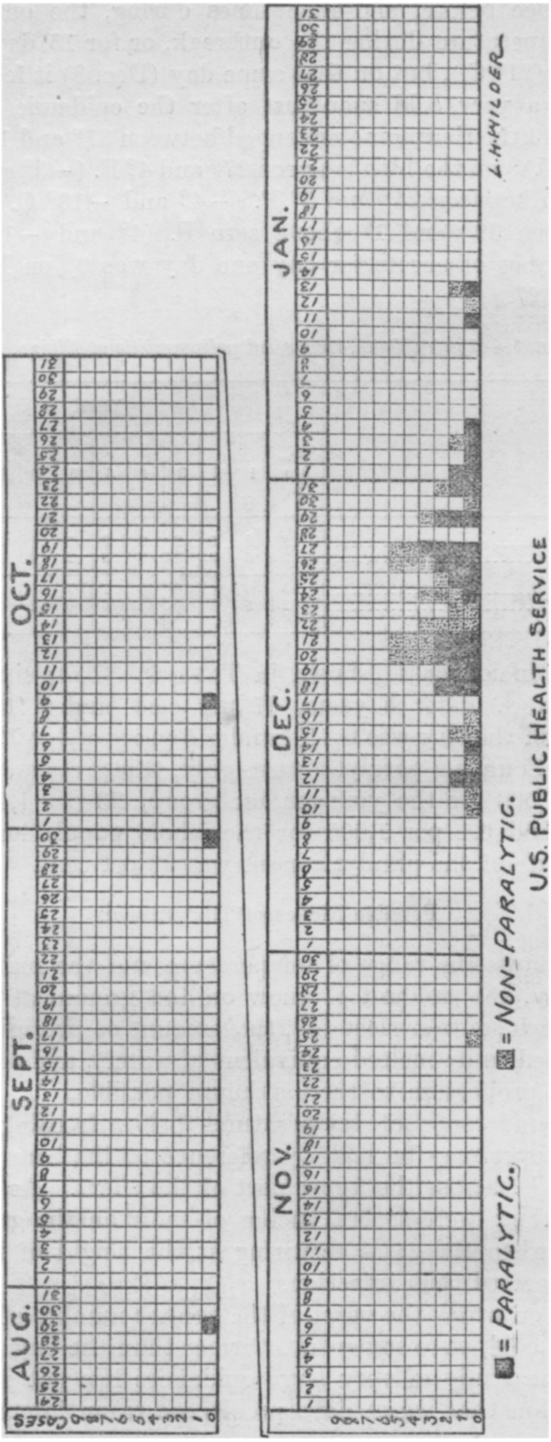
One case had its onset in spring, 4 had their onset in summer, and 3 in autumn. From these figures, 9 cases in 16 years in a population of 6,000, it would appear that a normal incidence of about 10 per 100,000, or 0.1 per thousand per year, is not an unreasonable estimate. This is, of course, higher than the reported incidence in any community during nonepidemic times, the New York City figures for 1909-1915 giving about 0.05 per thousand per year; but it is recognized that in the absence of an epidemic many cases are not reported, and only by such a canvass of the total population can the actual incidence be approximated.

No deaths from infantile paralysis were recorded for Elkins or Randolph County during the six years preceding 1916. Under the present system of recording deaths, a detailed analysis of the mortality statistics from other diseases could be of little value.

The Elkins Cases.

The onset of the first case of the real epidemic in Elkins (see Chart 1) was on November 25, the next on December 10, the midpoint on December 24, and the onset of the last case January 13. Three scattering cases were found to have occurred during the year previous to this, and these are included in the tables and charts, but of the real epidemic 33 cases occurred previous and 33 subsequent to December 24, with 4 cases on that date—70 in all. The maximum temperature was below freezing on November 15 and 25, December 13 to 15, 18, and 19, 29 and 30, January 11 and 12, 15 and 16, 19 and 26;

CHART 1:- POLIOMYELITIS. DATES OF ONSET OF CASES, ELKINS, W. VA. 1916-1917.



that is, twice before, and nine times during, the outbreak. The highest temperature during the outbreak, or for 15 days preceding, was 70° F. (21° C.), but on that same day (Dec. 8) it fell to freezing. There was a very cold snap just after the epidemic started. On December 13 the thermometer ranged between 31° and 14° F. (–0.5° and –10° C.); on the 14th between 27° and 4° F. (–3° and –15° C.); on the 15th between 24° and 8° F. (–4° and –13° C.); and on the 16th between 39° and 1° above zero F. (4° and –17° C.). The largest number of onsets for any one day was 6, on December 20, 21, 26, and 27.

TABLE 2.—*Age incidence, recognized poliomyelitis at Elkins, 1916-17.*

	Years.												Total.
	Under 1.	1	2	3	4	5	6	7	8	9	10	22	
Nonparalytic	2	2	5	2	9	5	5	1	3	0	0	0	34
Paralytic	3	6	9	4	7	2	2	3	1	0	1	1	39
Total	5	8	14	6	16	7	7	4	4	0	1	1	73
Fatal	2	1	3	0	1	0	0	2	1	0	1	0	11

The age incidence is shown in Table 2. Sixty-six per cent of the cases were under 5 years, 97 per cent under 10. The total population of the city was 6,340, under 5 years of age 776, and 1,586 under 10. Counting paralytic cases only, this gives a case incidence of 37 per 1,000 for the younger age group, 23 per 1,000 for those under 10, and 6.1 per 1,000 for the whole population. The case mortality rate of the paralytic cases was 28 per cent.

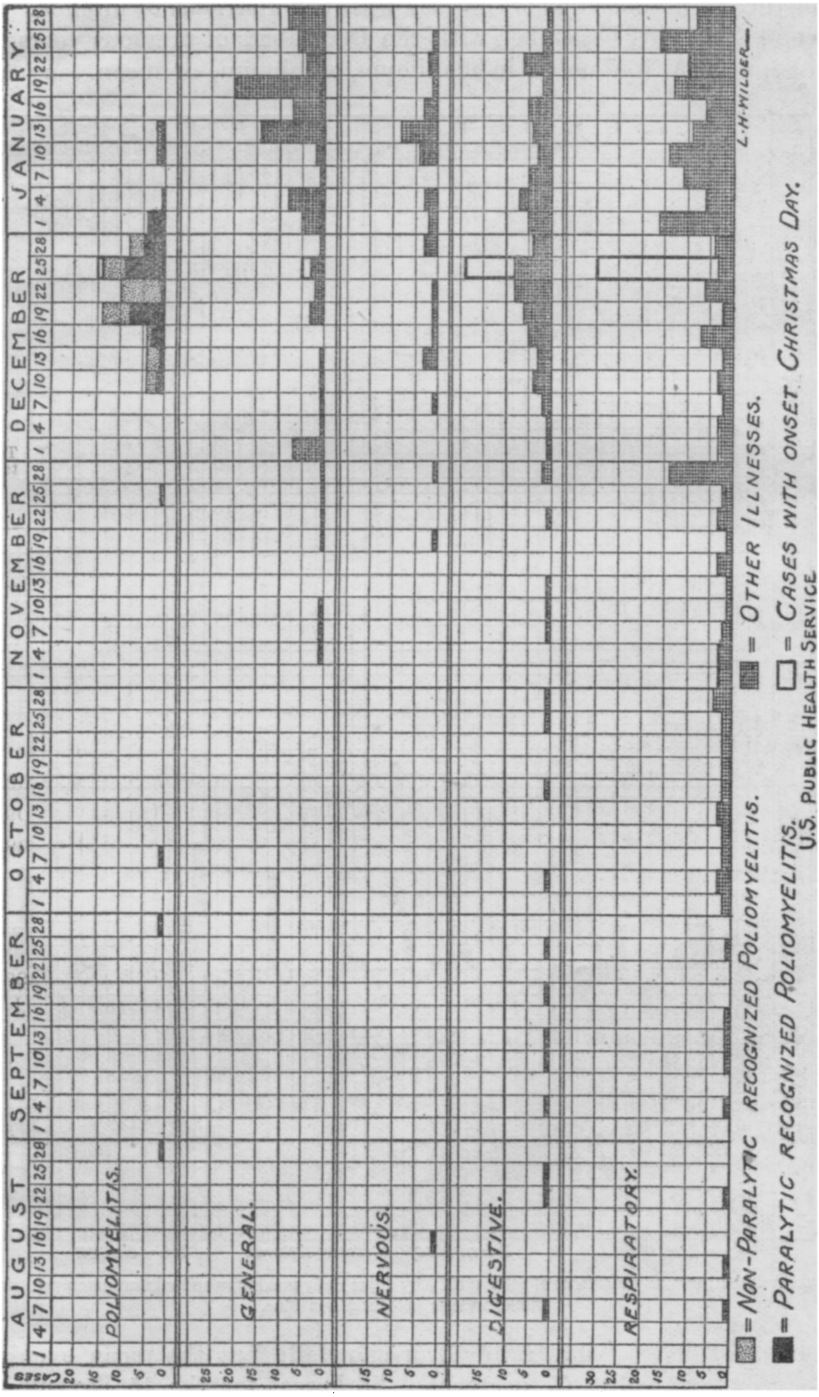
Possible Modes of Transmission.

Chart 3 shows the range of temperature and the number of cases on each day, the amount of snow on the ground at 8 p. m., the days on which snow or sleet fell, the amount of precipitation (snow being counted as its melted equivalent of water), and the amount of sunshine in proportion to the maximum possible.

The preponderance of temperatures below freezing during the epidemic proper may be seen by reference to the heavy line at the level of 32° F. across the upper part of the chart. As indicated on the chart, there were several inches of snow on the ground during the period when the cases occurring at the height of the epidemic probably received their infection.

The days on which the onset of the greater number of cases occurred, though cold, were somewhat warmer than the days just preceding; but the study of very large epidemics has not substantiated the impression that warm days predispose to an immediate attack

CHART 2.— POLIOMYELITIS AND OTHER DISEASES IN ELKINS, BY THREE-DAY PERIODS.



of the disease, such as is the case with the diarrheas of infancy. In comparing this December with the Decembers of previous years, no abnormality was noted in maximum, minimum, or mean tempera-

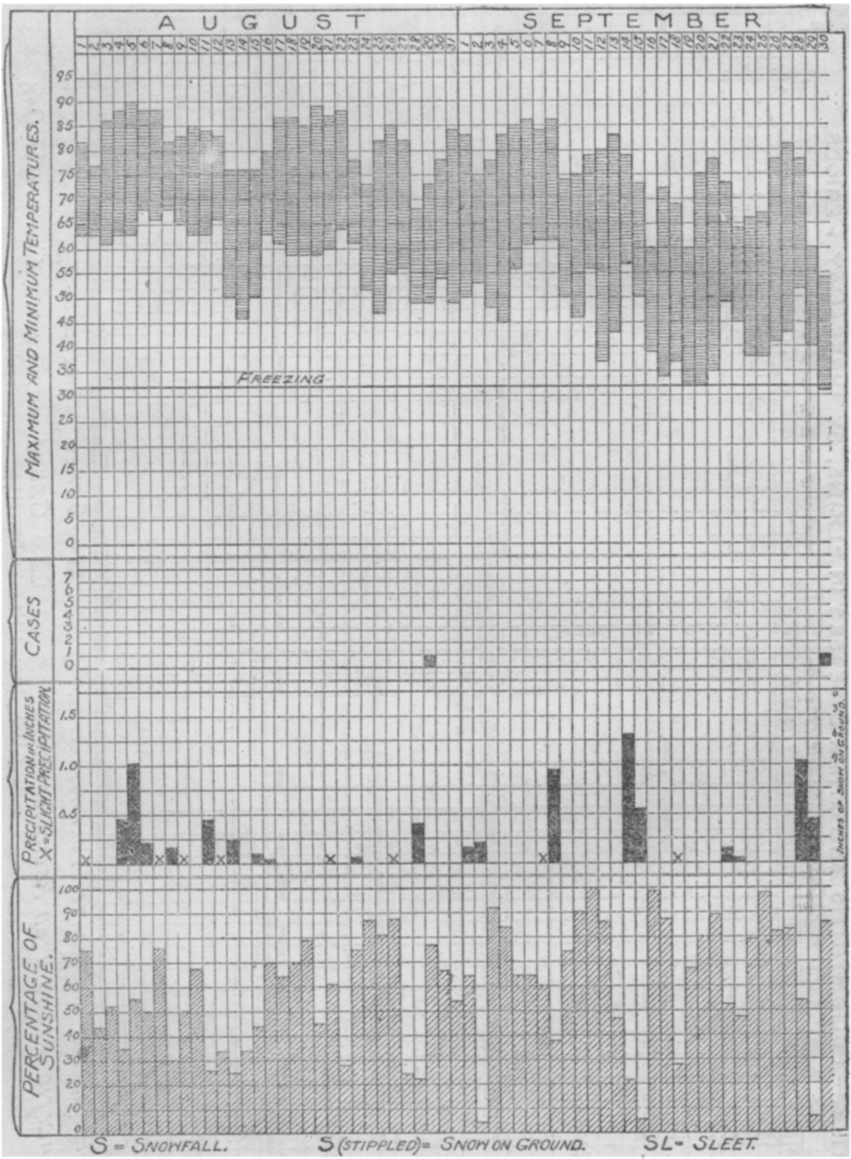


CHART 3, SECTION A.—Incidence of poliomyelitis by days; daily maximum and minimum temperatures; rainfall; cases; percent of sunshine.

tures, fogginess, wind, rainfall, or snowfall; but the mean evening humidity was low—67 per cent—the lowest for the 15 Decembers preceding being 74 per cent.

According to our observations of the surroundings of the patients, however, and information obtained from the families as to dustiness prior to onset, ordinary street or road dust is not likely to have played a part in causation of the disease. The morning humidity was normal for the month. Though poliomyelitis is universally recognized to be an infectious disease, and though the consensus of

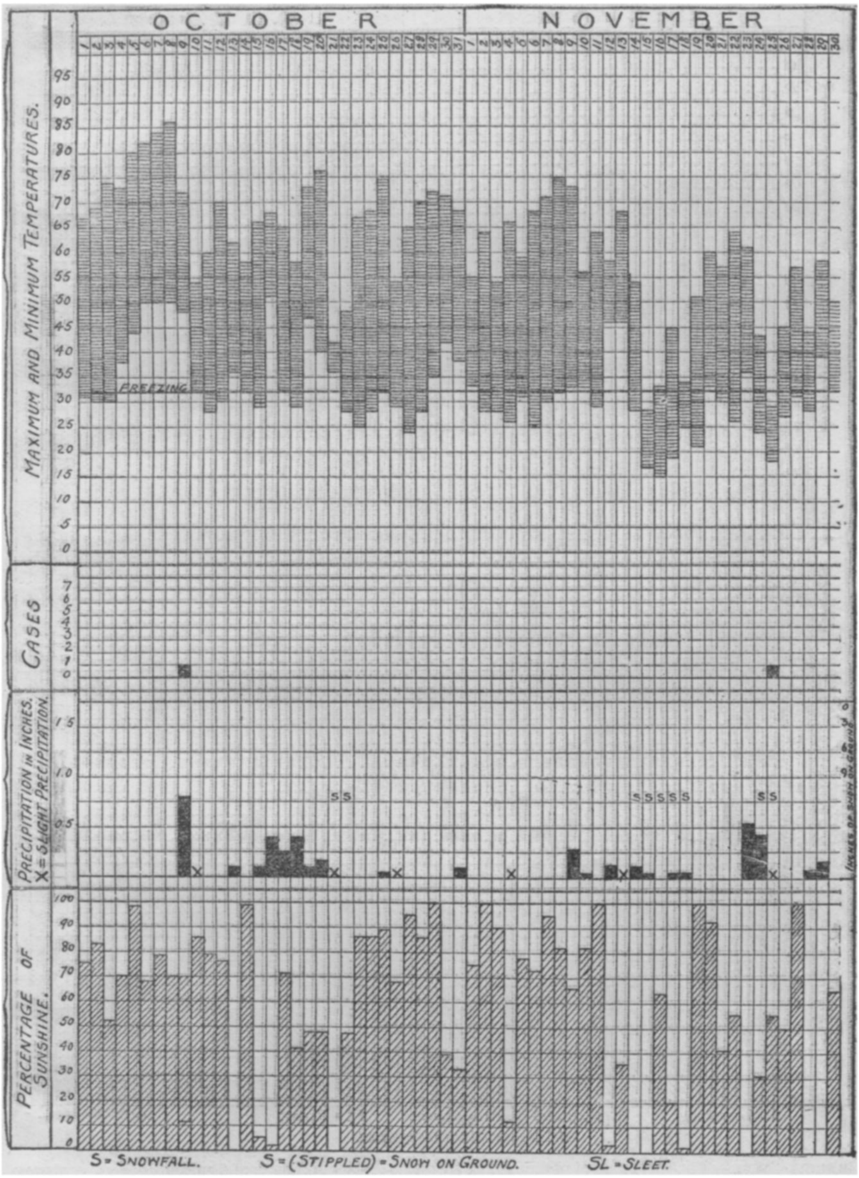


CHART 3, SECTION B.—Incidence of poliomyelitis by days; daily maximum and minimum temperatures; rainfall; cases; percent of sunshine.

opinion, now still further substantiated, ascribes its spread to contact, direct or indirect, it has been thought by some that peculiar meteorological conditions may predispose to the disease and determine an

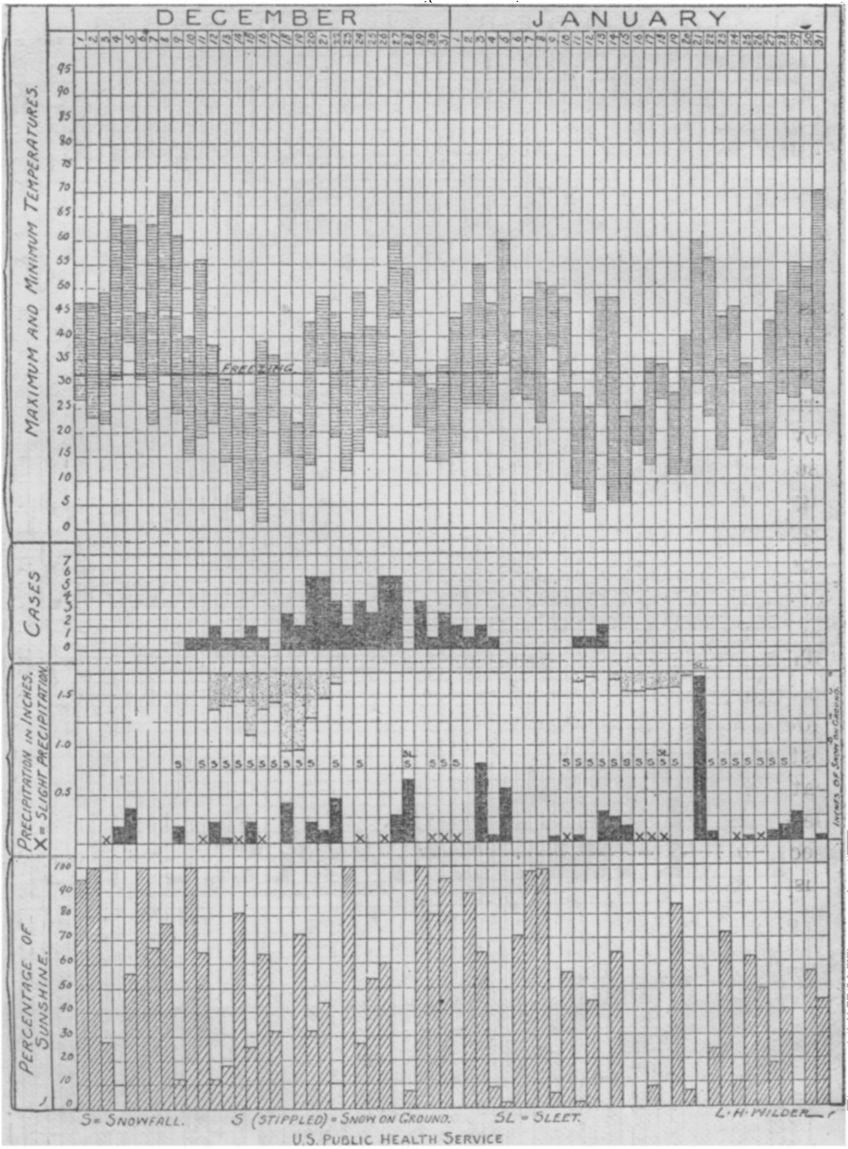


Chart 3, Section C.—Incidence of poliomyelitis by days; daily maximum and minimum temperatures; rainfall; cases; percent of sunshine.

epidemic, the germ itself being widespread in nonepidemic times. It has also been thought that attacks of rhinitis may allow the virus a nidus on the mucous membrane of the upper respiratory tract where it would otherwise be destroyed. The data collected for Elkins hardly give support to these ideas. One of the phases of this study to which particular care and much time was given was a house-to-house canvas of the entire population of the city for statistics as to age, sex, occupation, visits and visitors, and morbidity. In our canvas for all cases of illness occurring before, during, and after the epidemic, no undue prominence of coryza appeared, and the only abnormality noted in the weather conditions—an increased winter dryness—would tend to inhibit respiratory disorders.

Further, dust or meteorologic conditions could hardly have been factors of great importance, on account of the nature of the radial spread of the disease, as has often been pointed out in other epidemics. The outbreak subsided in Elkins at the same time that it was advancing in Grafton, and still later it spread to Fairmont, though all these places were experiencing weather conditions which at any one time were practically identical, but from first to last very variable, as regards humidity, temperature, dustiness, rains and snowfall, and wind. The 22 paralytic and 6 recognized nonparalytic cases in Grafton occurred from December 20 to January 29, with the height of the outbreak on January 7. The 15 paralytic and 4 recognized nonparalytic cases in Fairmont occurred from January 2 to February 11, with the height at this focus about the middle of January.[1] The weather at both these points during the outbreaks was fully as cold as that at Elkins.

Turning again to the temperature itself, below freezing 23 out of the 30 days preceding, and 31 out of the 34 days during the Elkins epidemic, and bearing in mind the 38 other winter outbreaks on record, most of them during much colder and some during extremely cold weather, it can be denied that summer insects play any necessary rôle in the spread of the disease. Careful watch was kept for such insects in Elkins. On five of the warmer days a few rather sluggish houseflies and other nonbiting flies were seen in residences. Mosquitoes were observed outdoors on three days. Stables, manure piles, and sunny walls were searched repeatedly for *Stomoxys calcitrans*, the biting stable fly, without success. Stable men affirmed, that no biting flies had been noticed since before Thanksgiving Day. It is inconceivable that such a sharp winter epidemic as this could have been caused by any of these insects.

As noted in previous epidemics, the incidence in the most cleanly homes, as well as in those less cleanly, is an effective argument against spread by insects commonly associated with filth, such as bedbugs and lice. This argument is especially potent here, for on the house-to-house canvass it can be affirmed that the proportion of scrupulously kept residences was as great among the infected as among the noninfected. Although bedbugs and lice may invade any home, if they were necessary carriers of the disease we should have a noticeably increased incidence in houses most likely to be frequented by them.

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[1] The data for the Grafton and Fairmont cases were obtained from the report of Dr. C. R. Weirich, director of the division of preventable diseases, West Virginia State Department of Health, to whom acknowledgment is also made for assistance during the epidemiological study in Elkins.

Among other things investigated, in accordance with the suggestion of Dr. Mark Richardson, of Massachusetts, were rats, but we found no evidence of an epizootic among rats, nor of particular association of cases with rats.

Though included in the schedule used in the investigation of each case, and made the subject of other more detailed investigation in certain instances, neither domestic animals nor ingested material could be incriminated as causing poliomyelitis in Elkins. Of the latter, milk and water were the two materials most susceptible of study; the histories elicited in each case compared with the general data for the city showed no relation to the disease. One of the babies—only 40 days old—had taken nothing but mother's milk up to the time of onset. Many other exclusively breast-fed cases have occurred in Elkins and elsewhere, but this is the youngest case on record, confirmed by histological findings at autopsy and by monkey inoculation.

As regards direct contact with recognized cases as a cause of the disease, our facilities for investigation were excellent, since by the house-to-house canvass all the cases which could, with reasonable degree of certainty, be called poliomyelitis were found. The list of these cases was carried to each infected house, and questions were asked concerning contact with each name on the list. Every case was visited more than once; some were visited several times. As a rule, about two hours were spent in each house on the first visit. On the blank schedule used particular attention was given to the acute clinical features and to contact, many other questions being asked to check and bring up from other angles answers to these questions. We can not refrain from commending the sane helpfulness of the physicians and people of Elkins. In a less homogeneous, or less thoroughly American, community, these studies would have been almost impossible. Even with this opportunity, possible direct contact—that is, contact with actual previous cases through which the disease might have been contracted—could be made out in only 26 per cent of the cases.

Quarantine of the cases could have had little to do with this low percentage of direct contacts, for the first nonfatal case was not reported until December 26, after practically all of the cases had received their infection. Indeed it was only with the knowledge of the presence of the epidemic that the early nonparalytic and fatal cases could be diagnosed in retrospect.

Indirect contact, through parents or acquaintances, occurred so frequently that analysis would be without avail. The incidence of the disease was heavy in Elkins, and the population mingled freely up to the height of the epidemic, when the disease began to be recognized. The greater part of the remainder of the cases were by this time in their incubation period. In all cases, however, repeated questions were asked concerning indirect contact, in the hope that additional opportunities for direct contact—that is, contact with patients themselves—might be remembered by the parents. Except in members of the same family, where frequently the dates of onset were so close as to suggest a common source of infection, the opportunities for contact with recognized cases when they occurred were usually not intimate, while in several cases mothers or other relatives of sick children were stated to have fondled children of other families who afterwards became sick.

On the face of these returns, therefore, granting that poliomyelitis is solely a human disease and is conveyed by contact (we have very slender evidence to lead us to believe otherwise), 74 per cent of the cases were occasioned by indirect contact through unrecognizable cases or through carriers. The other illnesses, which might have been unrecognizable poliomyelitis and which we shall speak of later, occurred for the most part in children. As regards possible carriers, it is interesting to note that in each of the two successful experiments on record (one [1] in New York and one [2] in Sweden) in which natural carriers were clearly demonstrated, the pooled nasal secretion included that from at least one adult. Furthermore, the usual history of indirect exposure obtained in the Elkins epidemic was through an adult.

It might well be that opportunities for direct infection presented themselves in Elkins which were not remembered by the parent, in stores, in street play, or at other times. In some of the rural cases studied, however, direct exposure to a recognizable case of the disease could not have taken place. On account of the severe weather the children had been kept close at home for weeks, and no other members of the family had been sick.

Any explanation of the mode of conveyance of poliomyelitis must assume that the relative susceptibility to the disease in all ages and in all places is low. Only a very small proportion of those exposed are attacked.

Clinical Features.

The general character of the epidemic, the age incidence, fatality rate, and clinical symptoms correspond with the New York 1916 epidemic, with the possible exception that some respiratory symptoms rather frequently accompanied the other acute symptoms. Forty per cent of the cases had onset with one or more remissions.[1]

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[1] Flexner, Clark, and Fraser: 1912—J. Am. M. Ass., vol. 60, pp. 201-202. (Jan. 18.)

[2] Kling and Pettersson; 1914—Deutsch. m. Wchnsch., vol. 40, p. 321. (Feb. 14.)

Incubation period.—Some data are afforded by this epidemic as to the incubation period of poliomyelitis. The periods formerly assigned, as low as one or two days, are now generally admitted to be too short. The possibility of simultaneous infection of two cases in one family or in close association by an adult carrier was not generally taken into account. We now recognize that two associated cases with onsets within a few days of each other probably have received their infection from the same source, instead of the later case from the former, the difference in time of onset being due to variation in incubation period. The figures based on the 1916 experience in New York and elsewhere have been given as 4 to 14 days.

This corresponds to the incubation period in artificially-infected monkeys; 4 days is about the minimum of highly virulent strains after having been passed through several generations of monkeys, while 14 days corresponds to the longer period, when a monkey is inoculated with material of lower virulence, for that species, direct from a human case. Incubation periods as long as 50 days, in monkeys, have been reported under these circumstances. The usual monkey inoculations giving the shorter incubation periods are made directly into the brain, and would be expected to produce the disease more quickly than infection by natural channels. If we may draw an analogy from rabies, a somewhat similar disease, the incubation period in the laboratory tends to be much shorter than in nature. We have one clear case in this epidemic giving an incubation period of at least nine days.

The child in question left Elkins after having been exposed directly to cases of the disease some days previous, and to possible carriers, of course, during the entire time in Elkins. He was out of contact with people from Elkins or other infected localities for nine days, when he was taken with the disease. This is more significant than the usual history of the sort, because, on account of the time of the year, poliomyelitis being in abeyance everywhere except in West Virginia, possible carriers from other localities can be more clearly excluded.

Diagnosis.—The question may be asked as to what constitutes a case. Our criterion for officially reporting a case, so that it would be quarantined, was that it should be of the paralytic type, showing, in addition to the acute symptoms, definite localized weakness (not a simple febrile weakness) or a clearly demonstrable disturbance of reflexes, a break in the reflex arc. Unless we base them on paralytic cases, statistics from different places are not comparable.

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[1] Public Health Reports, 1917: The diagnosis of poliomyelitis, vol. 32, No. 44, pp. 1831, 1833. (Nov. 2.)

There were 39 paralytic cases in Elkins, an incidence of 6.1 per 1,000 of population. But, even of these 39, we believe that the majority would not have been recognized or would have been counted as abortive in many localities. Of the 11 fatal cases, 6 were not recognized as such at time of death; of the nonfatal paralytic, 2 were not recognized until discovered in the house-to-house canvass, and 14 had such slight motor disturbance that recovery was complete in less than 3 weeks from onset.

Lumbar puncture would enable positive diagnoses to be made for quarantine purposes in such of the nonparalytic cases as are meningeal. But unless definite benefit to the patient can be promised from the lumbar puncture, relatively few parents will consent to the operation on the mild cases merely for diagnosis, and we believe that the proportion of abortive cases which can be officially diagnosed in this way will always be small. The fatal cases which occurred after our arrival in Elkins, though the clinical history was usually not clearly that of poliomyelitis (the symptoms being referred to the respiratory tract rather than to the musculature) were demonstrated positively by histological examination, and in two cases by monkey inoculation. This would indicate that very acutely fatal illnesses in children during the prevalence of poliomyelitis had best be considered cases of infantile paralysis, unless another diagnosis is clearly established.

In the diagnosis of the nonparalytic cases, we were sometimes assisted by the similarity of symptoms, if there was a paralytic case in the same family. In general, the nonparalytic cases which we recognized gave a history of fever with digestive disturbance, constipation and vomiting being two of the frequent symptoms, and either meningeal symptoms (hyperesthesia, pain on forward flexion of the neck, drowsiness, irritability) or a transient weakness out of proportion to the febrile disturbance. Headache, sweating, and retention of urine were often met with. A very common history in both paralytic and nonparalytic cases, when careful inquiry was made, was an onset with remissions; the child would be better for awhile and then worse again. We recognized 34 nonparalytic cases, seven-eighths as many as the paralytic. By far the greater part of the nonparalytic cases were not recognized until the house-to-house canvass was made, though some had been suspected previously.

One other question, to which we addressed ourselves, was whether poliomyelitis can occur with neither meningeal nor paralytic symptoms. In other words, are there many light cases of illness which we could not by any criteria, such as those just mentioned, class as poliomyelitis, and yet which are due to the same cause? It is obvious that a definite, proved answer to this question can hardly be given; if by a fortunate chance a positive monkey inoculation should be obtained from the nose and throat secretions of such a case, it would still be possible that the patient was a mere carrier, and that his symptoms had nothing to do with poliomyelitis; on the other hand, the neutralization test, to determine whether the blood serum destroys the virus, may give a positive result with those merely exposed to the disease, or may conceivably give a negative result with those who have suffered from a very light attack, since monkeys require a definite paralysis as a rule to become immune to the virus.

Our method of approaching this problem was to take a brief history of all cases of illness from August, 1916, to February, 1917, that could be remembered by the members of the household encountered in our house-to-house survey of every home in Elkins. Doubtless many were forgotten by the mothers or omitted for one reason or another; doubtless also those most vividly remembered were those occurring when everyone was on the lookout for poliomyelitis. But by a classification of these illnesses, and chronological comparison with the curve of the epidemic, it was thought that a tentative solution of our problem could be obtained.

At first, contact studies were made of the doubtful cases of poliomyelitis, but these grew so complex that the question of contact was finally confined to the 73 definite cases (39 paralytic and 34 nonparalytic).

Our 512 other cases of illness in the 1,370 families questioned were divided into four groups, respiratory, digestive, nervous, and general, according to their predominant symptoms. In the respiratory group, we placed those complaining principally of cough, cold, or sore throat; in the digestive, those with constipation, diarrhea, nausea, or vomiting; in the nervous, twitching or irritability; the general group comprised those with merely fever, malaise, skin, or urinary trouble, and such definite diseases as measles. The greater number of all these 512 cases were not sick enough to consult a doctor.

The result of the tabulation showed (see Chart 2), as was to be expected, that, in general, the most recent illnesses were best remembered. This is particularly the case with the respiratory, nervous, and general groups. On account of the likelihood of illnesses being caused by indiscretions of various sorts at Christmas, the cases with onset on that day were counted separately. But even disregarding these, a peculiar similarity is noted in the digestive cases and the poliomyelitis cases, as regards their time of onset; instead of being most numerous at the time of investigation (last of January and first of February), these illnesses, not diagnosable as poliomyelitis from the history, and some of them very trifling in character, show a tendency to group themselves around the time when poliomyelitis was prevalent. We were in entire ignorance of this result until the data were finally assembled and the charting was completed. In fact, we had supposed a priori that the nervous and respiratory cases would more naturally show a relation to the poliomyelitis; conscious or unconscious bias could hardly, therefore, have entered into the result. A summary of the chart appears in Table 3.

TABLE 3.—*Illnesses in Elkins as reported in house-to-house canvass, by 25-day periods.*

	Nov. 15-Dec. 9.	Dec. 10-Jan. 3.	Jan. 4-28.
Various illnesses	47	99	172
Digestive disturbances	8	54	34
Poliomyelitis	1	64	5

If histories of digestive disturbances had followed the chronology of the various illnesses, nervous, respiratory, and general, which were presumably not poliomyelitis, we should have had only 18 such for the 25-day period, December 10 to January 3, instead of 54. It may, therefore, be assumed that if our data are correct, about 36 digestive cases were unrecognized poliomyelitis, approximately as many as there were paralytic or recognized nonparalytic cases.

Thus we are led to believe it probable that other types of nonparalytic poliomyelitis than the meningeal do exist, and that in these other types symptoms referable in the first instance to the digestive tract are likely to be prominent. This is not saying, however, that the digestive tract is the portal of entry of the virus. Digestive symptoms in the paralytic cases come on only several days after exposure.

Administrative Control.

The tentative conclusion reached above, that nonparalytic cases of poliomyelitis occur, practically nonrecognizable on account of the absence of meningeal symptoms and their slight severity (frequently no doctor was called), is borne out by clinical observation of the recognized cases themselves. In the acute symptoms of poliomyelitis, as in the paralytic, we have all grades. It is a disease like scarlet fever as contrasted with measles, for example. We do have very light instances of the latter disease—measles—but they are the exception. A case of measles is usually definite and well marked. With scarlet fever and poliomyelitis, however, the symptoms in a considerable number of cases seem to trail off into nothing at all, to diminish indefinitely or without limit, as a mathematician would say. It is well known that cases of scarlet fever occur without eruption, but for administrative purposes we can regard only those which show some such definite diagnostic sign as the characteristic eruption or peeling. So in poliomyelitis we advised legal quarantine only for those in which we demonstrated definite paralytic phenomena, or the chemical and cellular changes in the fluid obtained at lumbar puncture.

What is to be done with the other cases, most of which do not now come to the attention of the health officer? In the first place, any numerical increase in the instances of digestive disturbance in children should be brought to the attention of the health officer for conference, particularly if slight meningeal signs are also present: disinclination to bend the body or neck forward, oversensitiveness to the touch, increased reflexes, irritability, drowsiness, restlessness, twitching or other nervous symptoms. Such an increase is said to have been noticed in Fairmont before the outbreak of recognized cases. We have previously mentioned the importance also of taking cognizance of deaths from acute illness in children, unless the diagnosis is very plain. Physicians will the more readily confer about these cases, when they realize that quarantine restrictions will not be oversevere. In our opinion, the suspicious and nonparalytic cases should be urged to adopt a voluntary quarantine, without placarding. An explanation of the dangers of the disease, the promise of immunity from paralysis if the case should actually be one of nonparalytic poliomyelitis, and a statement as to the reasonable methods of prevention will often, except in large centers of population, accomplish more than a shotgun quarantine.

At the same time, watch should be kept for confirmation of suspicions, and when a diagnosis is possible, the public should be informed as to measures of self protection, and the State authorities notified. At Elkins, before the first clearly paralyzed case occurred, there were 7 abortive cases, 2 fatal unrecognized cases, and 1 slight paralytic case, in which a definite diagnosis was afterwards possible solely on account of the residual reflex abolition on one side—10 cases which in many communities certainly would go unrecognized.

The data derived from this epidemic as to the period of infectiousness of the disease give no instance of infection occurring from exposure to a primary case longer than nine days after its onset. It would thus seem that two weeks from onset, or three weeks at the outside, is a reasonable period of quarantine. It must not be supposed that any quarantine of the recognizable cases, however perfect, will stop the spread of the disease. In the first place we have seen that direct contact even with abortive (nonparalytic), as well as with the paralytic, cases could account for only one-quarter of the outbreak. Further, it has been shown experimentally that the virus may be carried in the nose and throat as long as five months after the attack, an unthinkable period for quarantine. An additional period of, say 14 days, after quarantine, during which the patient and his family are kept from school or places of public assembly, is therefore wise. Our main reliance, however, is in general prevention of contact during the presence or threat of an epidemic. Parents should not fondle their children, lest the former should be unwittingly carriers of the disease. Business is not to be stopped, but unnecessary occasions of bringing people together in close contact should be warned against and prohibited as much as possible.

The interchange of nasal and throat secretions, which takes place when our neighbors talk in our faces, cough or sneeze, is to be brought to a minimum. Personal cleanliness and the avoidance of hand contaminations should be observed. It is recognized that these opportunities for infection can not be entirely extinguished in any independent community; but it is to be hoped that, just as reduction of anopheline mosquitoes below a certain number will stamp out malaria, so the general observance of the hygienic measures outlined above will tend toward preventing poliomyelitis.

Account must also be taken of possible infection through the digestive tract. If this takes place, and there is some reason to believe that it does, the pollution of articles of food and drink is probably not on so large a scale that the water or food supply of a whole community is infected from one case, but rather as a part of human contact, the articles placed in the child's mouth having been soiled, probably invisibly, by the secretions of some infected person in reasonably close contact with the child. This still means that the nose and mouth are the portals we must guard against infection and that we must bear in mind the general public, unrecognized cases and carriers, as more important factors than the officially reported cases themselves.

Summary.

To summarize the important points of our studies in West Virginia:

1. Modes of spread other than by human contact are improbable. Persons and not things should receive our attention.
2. Even including all the nonparalytic cases which we could recognize, direct contact with the cases themselves is responsible for only one-quarter of the incidence.
3. As passive carriers adults are probably more important than children.
4. There are probably many unrecognizable cases of the disease during an epidemic of poliomyelitis, about as numerous as the paralytic or the recognizable nonparalytic cases, and such cases are likely to have symptoms referable in the first instance to the digestive tract.
5. Quarantine of the case for two or three weeks is long enough; but another member of the family is just as likely to be dangerous as the patient, and general intermingling, particularly contact with children, should be prevented.